



## THE FORENSIC PANEL

TEL: 212 535 9286 FAX: 212 535 3259 MICHAEL WELNER, M.D., CHAIRMAN

October 29, 2021

James Loonam, Esq.  
Jones Day  
250 Vesey Street  
New York, New York 10281

Re: ***U.S. v. Robert T. Brockman***

Dear Mr. Loonam,

Pursuant to your request, I have conducted a forensic psychiatric evaluation of Robert T. Brockman. As noted in my report submitted to the Court on August 6, 2021, Mr. Brockman, 80 years old, is accused of directing and aiding a complex, intricate scheme, requiring multiple steps and dating back to 1981, to defraud the government of taxes on billions of dollars in capital gains income, as well as engaging in a complex scheme involving the purchase of tiered debt. The charges include tax evasion, conspiracy to commit tax evasion, willful failure to report offshore bank accounts, wire fraud, international money laundering, and obstruction of justice. The allegations in the indictment span decades and involve a labyrinth of entities, people, bank accounts, documents and global financial transactions.

In my capacity as a geriatric psychiatrist you engaged me as part of The Forensic Panel to examine medical issues reflecting on Mr. Brockman's competency to stand trial, along with Dr. Thomas Guilmette (neuropsychologist), Dr. Thomas Wisniewski (neurologist, neuropathologist), and Dr. Christopher Whitlow (neuroradiologist). My initial in-person evaluation of Mr. Brockman occurred on July 11, 2021. My report was filed on August 6, 2021.

At your request, I saw Mr. Brockman for a follow-up forensic evaluation in person on October 3, 2021. On the basis of my follow-up evaluation along with updated sources of information and collateral source input, I am addressing the following questions:

- 1) ***What diagnoses are reflected in the recent history and other testing data? Does the evidence reflect that Mr. Brockman is malingering cognitive incapacitation? Why or why not?***
- 2) ***Is Mr. Brockman able, given the nature of the charges against him, to assist his attorneys with relevant, requested facts, dates, and specifics?***
- 3) ***Does Mr. Brockman reflect the mental stamina needed for a courtroom trial on the charges he faces? Why or why not?***
- 4) ***Is Mr. Brockman able to assist his counsel in defending his case? Why or why not?***

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**US v. Brockman**

**DX-15**

Case No: 4:21-cr-0009

U.S. v. Robert T. Brockman  
*The Forensic Panel – Marc Agronin, M.D.*  
October 29, 2021

Page 2 of 22

What follows is a supplement and, when appropriate, an amendment to my August 6 report in this case. Please carefully review that report first in order to fully and accurately internalize the data collected and my analysis of the following, as this report aims to minimize repetition from the first report and rather aims to supplement it as an updated companion.

## **MORE RECENTLY REVIEWED SOURCES OF INFORMATION**

- 1) Sleep study, August 12-13, 2021
- 2) FDG PET scan report, August 24, 2021
- 3) EEG report, September 2, 2021
- 4) Medical records from Mr. Brockman, dates ranging from 1997-2014
- 5) Dr. Denney's interview notes with Kathy Keneally on June 14, 2021 and Pete Romatowski on June 15, 2021
- 6) Dr. Darby's interview notes with Dorothy Brockman
- 7) Medical records from Baylor College of Medicine for Drs. Pool and Smith through to August 18, 2021
- 8) Hospitalization records, September 15-18, 2021
- 9) Dr. Maria Ponisio's neuroradiology reports, September 1, 2, and 5, 2021
- 10) Peer conference call with Thomas Guilmette, Ph.D.,  
Bernice Marcopolous, Ph.D., James Seward, Ph.D., Elkhonon Goldberg, Ph.D.,  
and Michael Welner, M.D., September 30, 2021
- 11) Video recording and transcript of Mr. Brockman's interview with Thomas Guilmette, Ph.D., October 2, 2021
- 12) Video recording and transcript of Mr. Brockman's interview, October 3, 2021
- 13) Interview with Dorothy Brockman, October 3, 2021
- 14) Discussion with Frank Gutierrez, October 3, 2021
- 15) Dr. Thomas Guilmette's supplemental report, October 29, 2021
- 16) Medical record of Dr. Lai's examination, October 7, 2021
- 17) Video recording and transcript of Mr. Brockman's interview with Thomas Wisniewski, MD, October 17, 2021
- 18) Thomas Wisniewski, MD's supplemental report, October 29, 2021
- 19) Interview and transcript of Drs. Dietz and Denney's examination, October 20, 2021
- 20) Dr. Lai's interview with government prosecutors, September 8, 2021
- 21) Dr. Christopher Whitlow's supplemental report, October 29, 2021

## **UPDATES IN MEDICAL HISTORY**

Since the last report, the following additional medical events and testing have transpired:

U.S. v. Robert T. Brockman  
*The Forensic Panel – Marc Agronin, M.D.*  
 October 29, 2021

Page 3 of 22

DATE(s)	HISTORY
August 12-13, 2021	Sleep study: severe obstructive sleep apnea with improvement on CPAP. No REM sleep abnormal behaviors.
August 24, 2021	FDG-PET scan of Brain: “Mildly reduced uptake in the posterior temporal lobes and bilaterally in the parietal lobes. Slightly reduced uptake in the frontal lobes. Occipital uptake is reasonably good.” Findings “are mild, but very suggestive of neurodegenerative disease, particularly Alzheimer’s disease. Although statistically less likely, dementia with Lewy bodies or Parkinson’s disease with dementia can have a similar scan pattern. The markedly abnormal uptake on the prior Amyvid PET scan also somewhat favors Alzheimer’s disease over DLB/PDD.”
Sept 1, 2021	Interpretation of July 28, 2021 Amyloid PET / CT scan from Dr. Maria Ponisio (retained by government to review scans) stated that “this is a positive amyloid-PET study, indicating moderate to frequent beta-amyloid neuritic plaques.”
Sept 2, 2021	Electroencephalography (EEG) of brain waves: “mildly abnormal Video-EEG study characterized by diffuse slowing of the background, a non-specific indicator of global cerebral dysfunction.”
Sept 2, 2021	Dr. Ponisio interpreted the March 12, 2021 FDG-PET Scan of the brain, and opined that “the described pattern of hypometabolism can represent early Alzheimer’s dementia in the correct clinical setting. No significant decreased metabolic activity in the frontal and occipital lobes to suggest frontotemporal dementia spectrum or Lewy body dementia.”
Sept. 5, 2021	Dr. Ponisio interpreted the August 24, 2021 repeat FDG-PET Scan of the brain, and opined that “the described pattern of hypometabolism can be seen in early Alzheimer’s dementia in the correct clinical setting. When compared to prior examination, there is a mildly progressive decrease of metabolic activity in the compromised brain areas. No significant decreased metabolic activity in the frontal and occipital lobes to suggest frontotemporal dementia spectrum or Lewy body dementia.”
Sept. 13, 2021	Presented to Houston Methodist ER with a 2-3 week history of right elbow swelling after falling 3 weeks ago. He was described as “alert.” There was some swelling over the right elbow region. X-ray was negative for fracture. Diagnosed with olecranon bursitis likely due to trauma.
Sept 15 – 18, 2021	Hospitalization at Houston Methodist. Admitted with cough, confusion, lethargy, acute cystitis, fever and altered mental status, and discharged with diagnoses of urinary tract infection due to Klebsiella, acute toxic metabolic encephalopathy, and

U.S. v. Robert T. Brockman  
*The Forensic Panel – Marc Agronin, M.D.*  
 October 29, 2021

Page 4 of 22

	thrombocytopenia. He was treated with IV antibiotics and then switched to oral antibiotic upon discharge. The admission exam noted him to be confused and lethargic with decreased interactivity.
Sept 16, 2021	Brain CT scan: No acute findings. Microvascular ischemic changes of the white matter and diffuse volume loss.
October 7, 2021	Dr. Lai (neurology) saw Mr., Brockman for follow-up: “Clinical findings are consistent with Parkinson’s disease with associated dementia. His cognitive function has deteriorated since his last visit. . . . Neurological and cognitive examinations are worse compared to his last visit.” MoCA score was 13 out of 30.

## CURRENT MEDICATIONS

**Morning:** AZO cranberry urinary tract health 1 capsule in AM; Exelon patch 9.5 mg; Miralax capful; Carbidopa-levodopa 25-100 2 tablets; Bupropion SR 200 mg; Synthroid 75 mcg; Eliquis 2.5 mg; Stool softener 240 mg softgel; Vit D3 2000 IU 2 pills; Acidophilus softgel 1 pill

**Noon:** Carbidopa-levodopa 25-100 2 tablets; Cephalexin 250 mg

**4 pm:** Carbidopa-levodopa 25-100 2 tablets

**Night:** Trazodone 50 mg; Bupropion SR 100; Eliquis 2.5 mg; Rosuvastatin 5 mg; Quetiapine 25 mg

## COLLATERAL SOURCE INPUT

*Frank Gutierrez* works with Mr. Brockman on a daily basis as a personal aide and was also with him during our initial evaluation on July 11, 2021. He described Mr. Brockman’s current baseline state as being frequently confused and needing cueing and assistance with most daily activities including selecting and putting on clothes, preparing foods, attending to his hygiene, running his daily schedule, and ambulating safely.

He noted that Mr. Brockman has continued to require significant close supervision and hands-on assistance with daily activities as he has over the past year, but his overall physical and mental conditions have declined even more over the course of his recent hospitalizations. Mr. Brockman will sometimes start packing a briefcase and try to leave to “go to work.” When he is reminded that he is at home and not working at his office any longer, Mr. Brockman will sometimes get angry and agitated verbally.



U.S. v. Robert T. Brockman  
*The Forensic Panel – Marc Agronin, M.D.*  
October 29, 2021

Page 5 of 22

According to Mr. Gutierrez, Mr. Brockman has been frail and unsteady and has had several recent falls, including one in late August in which he hurt his right elbow (see notes above on ER visit on September 13) and then another one after returning from the hospital later in September in which he sustained a mild bump to his head and some redness on his shoulder.

Mr. Gutierrez stated that Mr. Brockman is not preparing snacks like sandwiches independently, but only occasionally and with direct supervision. In mid- September, related by Mr. Gutierrez, Mr. Brockman became less coherent, weak, less alert, had decreased appetite and a glazed look on his face, and developed a fever up to 102.3. This decline necessitated a hospitalization on September 15<sup>th</sup>. Since discharge Mr. Brockman reportedly remains quite weak and in steady, with sleep OK and appetite “not great.”

***Dorothy Brockman*** also described the same state of mind and events for her husband as did Mr. Gutierrez. She stated that her husband has been more confused since returning from the hospital, and that the urinary tract infections have taken a general toll on his health. According to Mrs. Brockman, his cognition is better in the morning; but he gets sleepy in the afternoon and more confused. He asks his caregiver to pack his bag to go somewhere, and he is not always certain he is in Houston as he is asking to go to Houston. She related that her husband gets annoyed with a “scary deer in the headlights look” when reminded that he is already in Houston at his home.

Mrs. Brockman added that his appetite has considerably diminished. His function has declined in that he can’t walk alone as “he would get lost” and is very unsteady, and they are fearful of falls.

At times, Mr. Brockman reportedly puts pants on his arms or shirts on legs and doesn’t know which pants leg to use. She stated that he needs assistance with dressing and showering. Moreover, she indicated that he doesn’t know phone numbers of anyone and cannot access or process his email. She observed that he seems more depressed at times especially when hearing bad news from Dorothy, but he does not retain the information well.

According to Mrs. Brockman, he is still “sweet and loving” and knows some people by face but doesn’t always get names right. She added that he is somewhat disinhibited in front of people with poor judgment, such as sometimes saying “bizarre” things to people such as telling one friend he hasn’t had sexual relations in 25 years, which isn’t true. He talks about Reynolds all the time. Dorothy confirmed that they moved into their home many months ago and not “two weeks ago” as Mr. Brockman has stated.

***Neuropsychological Testing:*** Dr. Thomas Guilmette conducted repeat neuropsychological testing on Mr. Brockman on October 2, 2021. Based on five measures

U.S. v. Robert T. Brockman  
*The Forensic Panel – Marc Agronin, M.D.*  
October 29, 2021

Page 6 of 22

of the validity of his performance, Dr. Guilmette concluded that Mr. Brockman's neuropsychological test scores were valid and did not reflect evidence of malingering.

On the RBANS, which is a global measure of neuropsychological functioning, Mr. Brockman performed quite poorly with an overall score below the 1<sup>st</sup> percentile. This testing exhibited severe impairment of short-term memory and overall, no significant changes since testing in July of 2021. Dr. Guilmette concluded that "the RBANS continue to reveal significant impairments in multiple cognitive domains consistent with dementia."

Measures of attention, mental processing speed, and working memory revealed significant difficulties. Mental and physical fatigue limited his capacity to "maintain his focus and respond effectively to what is transpiring around him."

Mr. Brockman's "problem-solving approach was disorganized and random," consistent with his performance in July.

His language abilities were a relative strength on the RBANS and other tests, with some improvement since July in "auditory comprehension and inference" on one test.

Dr. Guilmette noted overall some variability in test scores compared to July, with it "generally unchanged" in the balance (specifically with declines in working memory and processing speed) with Mr. Brockman continuing to show "episodic confusion and confabulation" but "somewhat more anchored in reality in October during his confused episodes." He stated that the overall decline in Mr. Brockman's condition since testing by Dr. Michele York in March of 2019 is reflected in the fact that "Mr. Brockman appears to be thinking more slowly and taking longer to process information."

**Other Testing:** An EEG conducted at the government's request on September 2, 2021 showed diffuse slowing of brain waves and was interpreted by Dr. Brandy Ma as indicating "global cerebral dysfunction." This finding does not reflect delirium at the time of the testing.

A PET scan performed at the government's request on August 24, 2021 using radiolabeled glucose (fluorodeoxyglucose or FDG) to show brain metabolic functioning was interpreted by Dr. Ronald Fisher as showing "Mildly reduced uptake in the posterior temporal lobes and bilaterally in the parietal lobes. Slightly reduced uptake in the frontal lobes," suggestive of a neurodegenerative disease such as Alzheimer's disease, or alternatively, dementia with Lewy bodies or Parkinson's disease with dementia. Dr. Ponisio opined that "the described pattern of hypometabolism can be seen in early Alzheimer's dementia in the correct clinical setting. When compared to prior examination, there is a mildly progressive decrease of metabolic activity in the compromised brain areas. No significant decreased metabolic activity in the frontal and occipital lobes to suggest frontotemporal dementia spectrum or Lewy body dementia."

U.S. v. Robert T. Brockman  
*The Forensic Panel – Marc Agronin, M.D.*  
October 29, 2021

Page 7 of 22

## MENTAL STATUS EXAMINATION

I met with Mr. Brockman over the course of several hours on the afternoon of October 3, 2021 in a conference room at the Houston office of Jones Day. His personal aide Frank Gutierrez escorted Mr. Brockman into the room holding on to his arm, but then left the room, and I interviewed Mr. Brockman alone.

Mr. Brockman presented as an elderly man with an unsteady and mildly shuffling gait, and appeared as if he would fall over without his aide holding onto his arm. He had a mild pill-rolling tremor observed intermittently in his hands. He had a slightly masked facial expression without any clear emotional expression and made good eye contact. He was casually dressed and nicely groomed. He was quiet and passive with a cooperative and friendly demeanor, and answered questions with a somewhat muted tone of voice.

Mr. Brockman did not remember who I was from the last interview. During our initial introductions he appeared to understand that I was there to evaluate his "capacity" for his trial. I queried him about what that meant, and he provided a vague response:

DR. AGRONIN: And when you say "capacity," what do you mean by "capacity"? What does that mean, exactly?

MR. BROCKMAN: I guess probably the first piece of capacity is that you've got to be in a situation where you show up for work and contribute, and the company involved is 53 years old. It has a lot of complexity to it, since it's a computer-based software system.

DR. AGRONIN: And so what does "capacity" mean, exactly? What type of capacity? How would you explain it?

MR. BROCKMAN: I don't know that I can explain it from a legal standpoint.

DR. AGRONIN: Well, you told me a few minutes ago that this part of the -- your legal situation is looking at your capacity, so I guess I wanted to understand from you, what exactly does "capacity" mean in this situation? How will your capacity affect this case?

MR. BROCKMAN: Well, I understand there's at some point to be a hearing and the judge will rule as to whether or not I have capacity or not.

DR. AGRONIN: Okay.

MR. BROCKMAN: If I have capacity, it will be my job to assist our lawyers.

U.S. v. Robert T. Brockman  
*The Forensic Panel – Marc Agronin, M.D.*  
October 29, 2021

Page 8 of 22

DR. AGRONIN: What would it mean to have capacity to assist the lawyers? What does that mean exactly?

MR. BROCKMAN: I'm not... from a legal standpoint, I'm not familiar with the definitions of terms.

DR. AGRONIN: Okay.

MR. BROCKMAN: I think probably at a most basic level, "capacity" means that I can come to work and probably work all day long with lawyers.

DR. AGRONIN: What type of work do you do now?

MR. BROCKMAN: Right now, I'm retired.

DR. AGRONIN: Okay. What would your lawyers need you to do for them?

MR. BROCKMAN: I don't know.

Although he was aware of the general purpose of the exam, it took several reminders throughout the examination for him to remember my name and what my role was. He was eventually able to recall my name by the end of the interview.

He was attentive at times, but on several occasions appeared to stare off into the distance with a latency to his response. At several points he digressed into talking about work, unrelated to my question or the topic at hand:

DR. AGRONIN: Okay. In general, how do you feel that you can help your attorneys?

MR. BROCKMAN: Well, they have more than several interesting lawsuits underway involving big dealerships, groups of dealerships and some very complex software and their demands that we do what needs to be done to the software to make it exactly the way they like it.

DR. AGRONIN: And is that what you feel you can help them with?

MR. BROCKMAN: Yeah. Because I've been long-term involved with this particular account and I know the players inside pretty well and the guys in this dealership are basically big bullies and that they insist that such and such be done to the software or not be done to the software and they want to dictate, much as you would do if you had an in-house software department. Well, we don't have any provisions for that,



U.S. v. Robert T. Brockman  
*The Forensic Panel – Marc Agronin, M.D.*  
October 29, 2021

Page 9 of 22

and so therefore -- and these guys show up about once a year and they have a known pattern. They say hooray, glad everything's looking up, you know, we're going to make great progress this year, so forth and so on, and then what happens, it's like you started the record over again for an hour, two hours, however long it takes to kind of grind through their litany. At the end of the day, they're offering nothing, and therefore we're going to do nothing, and those are issues which are really... they're sales issues, in one regard, and then what do you do with a large, cantankerous customer that's trying to throw its weight around?

DR. AGRONIN: So how can you help your attorneys with these cases?

MR. BROCKMAN: Well, right now my attorneys understand zero about this part of our business, and they also know nothing about the history with this particular account, which has been long and bloody. If we were to build this piece of software the way this rambunctious dealer wants, conceivably could anybody else want it or would they have their own way of doing things.

DR. AGRONIN: And is this relevant to your case as well?

MR. BROCKMAN: Yeah, I think so.

DR. AGRONIN: How would it be relevant to your case?

MR. BROCKMAN: Well, the results of my case will have a material impact on the operation of Reynolds and it would be a typical move by a manufacturer such as Reynolds to give in and do, you know, whatever somebody wants, which in the business Reynolds is in, that is a sure key to default.

Mr. Brockman was not able to provide any insight into why his capacity is being questioned, and was not able to tell me that he had previous assessments and diagnoses of Parkinson's disease and dementia:

DR. AGRONIN: Do you have any underlying conditions that are causing you to walk slowly?

MR. BROCKMAN: The general overall balance.

DR. AGRONIN: Okay.

MR. BROCKMAN: And a good healthy fear of falling again.

DR. AGRONIN: But no diseases or medical conditions that are also causing problems?

U.S. v. Robert T. Brockman  
*The Forensic Panel – Marc Agronin, M.D.*  
October 29, 2021

Page 10 of 22

MR. BROCKMAN: Well, I have a disease which is... I don't know the name of it. It's fairly well-known to treat the kind of injuries I got going on inside me.

DR. AGRONIN: Do you have any type of medical conditions or diseases that are affecting your capacity?

MR. BROCKMAN: Yes.

DR. AGRONIN: Can you tell me what that would be.

MR. BROCKMAN: Well, this particular disease, the first thing that it does to you is it screws up your memory.

DR. AGRONIN: Okay.

MR. BROCKMAN: This is very frustrating to me because I had, at one point in time, a very good memory.

DR. AGRONIN: Yeah.

MR. BROCKMAN: No longer.

DR. AGRONIN: Have you ever had an evaluation for your memory before, aside from what I'm doing here?

MR. BROCKMAN: Yes.

DR. AGRONIN: How long ago was that.

MR. BROCKMAN: I would think the first one was four or five years ago, and it came out of an annual exam.

DR. AGRONIN: And did they give you a diagnosis or what did they tell you exactly?

MR. BROCKMAN: Well, first of all, they told me they wanted to take more tests.

DR. AGRONIN: To take more tests?

MR. BROCKMAN: Yeah.

DR. AGRONIN: Okay.

U.S. v. Robert T. Brockman  
*The Forensic Panel – Marc Agronin, M.D.*  
October 29, 2021

Page 11 of 22

MR. BROCKMAN: But this disease that I've got is incurable, progressive, attacks your memory, and also your ability to think, do things, like make computations.

DR. AGRONIN: Does it affect your body too?

MR. BROCKMAN: Yeah.

DR. AGRONIN: Do you know the name of any of these conditions that you have?

MR. BROCKMAN: I cannot remember but again, I rely on the fact that medical records are what's really... I believe that is the truest statement of what's going on inside me, not anything I got to say.

He was overall a poor historian, unable to recall recent events accurately, and telling me that he only recently moved into his house 4 – 5 weeks ago when in actuality it had been seven to eight months ago. Mr. Brockman occasionally demonstrated some general information about his criminal case, noting that “the IRS has taken the position that just because the trust earned money, that means I earned it too.” Overall, he had impoverishment of thought, lacking spontaneous details or elaboration except for when he digressed into talking about his previous work.

His affect was somewhat flat with a restricted range of emotional expression. He reported feeling nervous, angry over his case and somewhat depressed, like a “4 to 5” on a scale where “a 9, for me, would be sitting in the car and crying.” He denied suicidal ideation. He did not report any hallucinations or verbalize any delusional thinking. Mr. Brockman’s responses were generally superficial, vague with minimal detail and a lack of logical flow of abstract thinking:

DR. AGRONIN: Yeah. What have you been doing for your own case lately?

MR. BROCKMAN: I think mainly it's been going to the doctor, going through tests, you know, turning over medical information, trying to, you know, follow up what my doctors want me to do so I can recover at full strength. I've never been sick like this, never before.

DR. AGRONIN: Are there things you can do to get your health better?

MR. BROCKMAN: Yeah. I call it walking, I call it treadmill. It's called, you know, little two-pound dumbbells.

DR. AGRONIN: Have you been doing that?

MR. BROCKMAN: More. Still not perfect.

U.S. v. Robert T. Brockman  
*The Forensic Panel – Marc Agronin, M.D.*  
October 29, 2021

Page 12 of 22

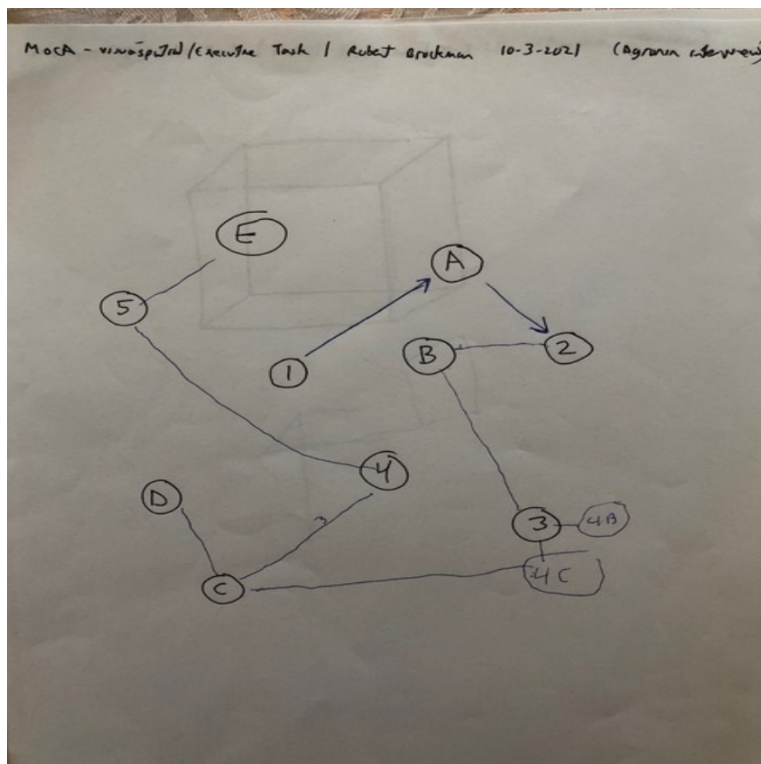
DR. AGRONIN: Yeah.

MR. BROCKMAN: But it's -- fortunately it's very simple and that's probably been very fortunate, the fact that it's simple, is what I was able to tell my wife about it and she understood it and so now she's the one that's after me.

His language function was grossly intact as he was able to understand my questions and respond in a coherent way.

On the **Montreal Cognitive Assessment**, Mr. Brockman scored 14 out of 30, in the moderately impaired range, slightly higher compared with his last score in July, 2021 of 9 out of 28.

**Visuospatial / Executive Tasks:** Impaired (1 out of 5 points): Mr. Brockman was able to attend to the trail-making, cube copy and clock drawing tasks, but spent time with each providing disorganized, distorted, perseverative and incorrect responses, as seen in the figures below:

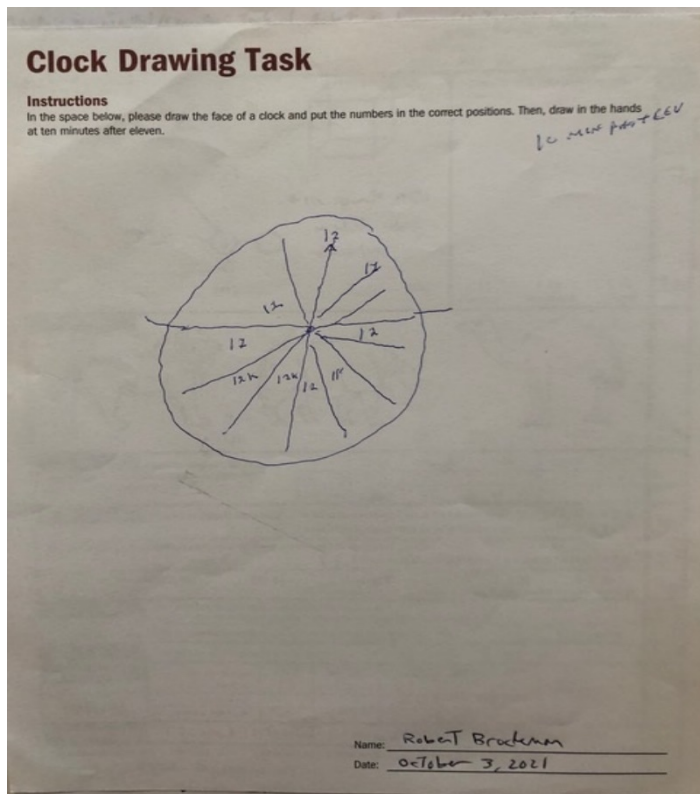
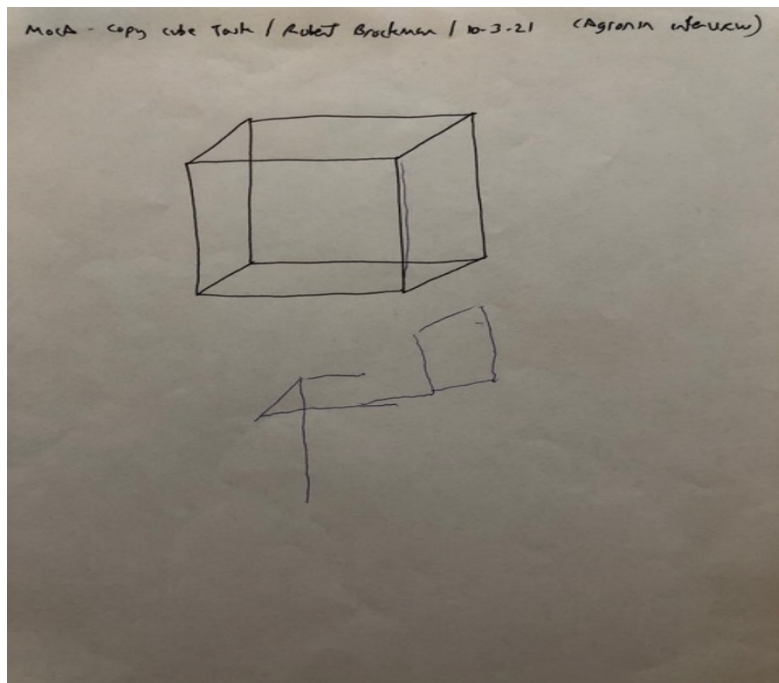


U.S. v. Robert T. Brockman

*The Forensic Panel – Marc Agronin, M.D.*

October 29, 2021

Page 13 of 22





U.S. v. Robert T. Brockman  
*The Forensic Panel – Marc Agronin, M.D.*  
October 29, 2021

Page 14 of 22

***Naming:*** Intact (3 out of 3 points): Mr. Brockman was able to easily name and then recall the names of three animals (lion, rhinoceros and camel).

***Memory / Delayed Recall:*** Impaired (0 out of 5 points): Mr. Brockman had significant difficulty repeating correctly a list of five words over two trials, and repeatedly mispronounced one word. He could not recall any of the five words after about 5 minutes.

***Attention:*** Impaired (4 out of 6 points): Mr. Brockman was able to repeat short digit lists forwards and backwards, and to tap his hand when the letter “A” was read out of a series of letters. He could only get one correct answer with subtracting the number 7 from 100 in a sequence of subtractions.

***Language:*** Impaired (1 out of 3 points): Mr. Brockman was able to repeat one out of two sentences, and struggled to create a list of words that begin with the letter “F.”

***Abstraction:*** Intact (2 out of 2 points): Mr. Brockman was able to correctly interpret two-word similarities.

***Orientation:*** Impaired (3 out of 6 points): Mr. Brockman was disoriented to time, telling me incorrectly that it was Thursday, October 22<sup>nd</sup>, 2022 (actual date was Sunday, October 3, 2021). He knew he was in his attorney’s offices in Houston but could not recall the name of the firm.

## FORENSIC PSYCHIATRIC ASSESSMENT

*1) What diagnoses are reflected in the recent history and other testing data? Does the evidence reflect that Mr. Brockman is malingering cognitive incapacitation? Why or why not?*

Mr. Brockman suffers from the following diagnoses:

1. **Parkinson’s disease dementia** (G20) with associated depression (F06.31), anxiety (F06.4) and psychosis (F06.2); more recently he has also demonstrated mild behavioral disturbances (F02.81)
2. **Possible comorbid Alzheimer’s disease** (G30.9)
3. **Apathy** (ICD-10 does not include a specific code for apathy)
4. **Insomnia with obstructive sleep apnea** (780.51)
5. **Possible REM sleep behavior disorder** (G47.52)

As noted in my initial report, Mr. Brockman has a well-established history of motor changes as well as neuro-imaging showing cerebral dopamine deficiency which has been

U.S. v. Robert T. Brockman  
*The Forensic Panel – Marc Agronin, M.D.*  
 October 29, 2021

Page 15 of 22

diagnosed by multiple neurologists as Parkinson's disease (PD). He continues to show these symptoms with some degree of motor response to levodopa-carbidopa.

Mr. Brockman has well-established progressive neurocognitive impairment which meets all criteria for Parkinson's disease dementia (PDD), including underlying PD with onset prior to the onset of neurocognitive impairment, global cognitive deficiency demonstrated on previous and current neuropsychological testing across at least two major cognitive domains (and in his case, across all cognitive domains), and resultant impairment of daily life reflected in his poor and worsening functional impairment.

The degree of Mr. Brockman's cognitive and functional impairment severely impairs his daily life and goes well above and beyond the threshold of mild cognitive impairment due to PD. As Aarsland and colleagues state unequivocally in their review paper on cognitive decline in Parkinson disease, "In PDD, but not in PD-MCI, the cognitive deficits are severe enough to impair daily life (for example, social and occupational functioning, and personal care), independently of the impairment ascribable to motor or autonomic symptoms."<sup>1</sup>

Mr. Brockman's neuroimaging includes both a positive amyloid scan indicating significant amyloid plaque in his brain, as well as a pattern of metabolic impairment on FDG-PET scan, consistent with findings in Alzheimer's disease (AD). These findings, coupled with his progressive neurocognitive impairment, suggest the likelihood that Mr. Brockman is suffering from comorbid AD, which can be seen in concert with PD and which predicts "future cognitive decline and dementia in PD."<sup>1, 2</sup>

The strongest piece of evidence indicating AD is the combination of the positive amyloid-based scan and metabolic / FDG-PET scan, both of which were read as indicative of AD by the neuroradiologist who did the initial interpretations as well as by Dr. Whitlow and Dr. Ponisio.

In one study that looked at the both the amyloid-based PET scans (using a tracer called PIB) and the metabolic / FDG-PET scans of 101 individuals which were then compared to autopsy findings, the authors found nearly perfect detection of AD when both scans were positive and thus congruent with one another. As the researchers stated:

"In summary, both amyloid and FDG PET showed high accuracy in detecting AD pathology, although PIB had higher sensitivity and negative predictive value, particularly in early disease stages. PIB and FDG performed comparably in identifying AD as the primary

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<sup>1</sup> Aarsland D, Creese B, Politis M, et al. **Cognitive decline in Parkinson disease.** *Nat Rev Neurol.* 2017;13(4):217-231.

<sup>2</sup> Palermo G, Tommasini L, Aghakhanyan G, Frosini D, Giuntini M, Tognoni G, Bonuccelli U, Volterrani D, Ceravolo R. **Clinical Correlates of Cerebral Amyloid Deposition in Parkinson's Disease Dementia: Evidence from a PET Study.** *J Alzheimers Dis.* 2019;70(2):597-609.

U.S. v. Robert T. Brockman  
*The Forensic Panel – Marc Agronin, M.D.*  
 October 29, 2021

Page 16 of 22

etiologic pathology underlying clinical impairment. When PIB and FDG were congruent, sensitivity, specificity, and total accuracy approached 100%.”<sup>3</sup>

Mr. Brockman has congruent amyloid PET and metabolic / FDG PET scans, indicating the strongest possible biomarker evidence of underlying AD.

PD is an unfortunately common neurological disorder, affecting approximately 1 million Americans with over 70% to 80% developing dementia over the course of the disease.<sup>4, 5, 6</sup> It is progressive and irreversible.

AD is vastly more common, affecting nearly 6 million Americans and defined by the presence of progressive and irreversible dementia. My own examination of Mr. Brockman, compared to my exam in July, 2021 and coupled with collateral reports from his personal aide, wife, and neuropsychological findings, reflects ongoing progressive neurocognitive impairment as seen in both PDD and AD, with evident worsening in executive function / abstract thinking, mental processing speed, and memory function. The decline in his overall neurocognitive, physical and functional state is all consistent with the known courses of both PD (and PDD) and AD.

Mr. Brockman has had several recurrent bouts of delirium this year, each one in the setting of a urinary tract infection and involving more acute confusion over his baseline impairment, weakness, lethargy, decreased attention and alertness, and agitation.

In the July 2021 examination, I noted that Mr. Brockman frequently digressed into nonsensical, illogical and at one point paranoid responses to questions that indicated his attention and train of thought were impaired. This presentation was indicative of residual delirium. Since that time Mr. Brockman has had another bout of delirium, but with resolution of the infection and less disorganized mental status since then according to his personal aide and wife.

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<sup>3</sup> Lesman-Segev OH, La Joie R, Iaccarino L, et al.: **Diagnostic Accuracy of Amyloid versus <sup>18</sup>F-Fluorodeoxyglucose Positron Emission Tomography in Autopsy-Confirmed Dementia.** *Ann Neurol.* 2021 Feb;89(2):389-401.

<sup>4</sup> Marras, C., Beck, J. C., Bower, J. H., et al., on behalf of the Parkinson's Foundation P4 Group (2018). **Prevalence of Parkinson's disease across North America.** *Npj Parkinson's Disease*, 4(1), 1–7.

<sup>5</sup> FAQs: Dementia and Parkinson's. Parkinson's Foundation website. Accessed October 10, 2021 at: [https://www.parkinson.org/Understanding-Parkinsons/Symptoms/Non-Movement-Symptoms/Dementia/FAQs?utm\\_source=google&utm\\_medium=adgrant&utm\\_campaign=&utm\\_term=incidence%20of%20parkinson%27s%20disease&gclid=CjwKCAjwh5qLBhALEiwAloods9Nez7LkaBBZbGqc2SMv-rsXKgmVqrmD84SoRt5op-KG-8ABkBoIBoCxakQAvD\\_BwE](https://www.parkinson.org/Understanding-Parkinsons/Symptoms/Non-Movement-Symptoms/Dementia/FAQs?utm_source=google&utm_medium=adgrant&utm_campaign=&utm_term=incidence%20of%20parkinson%27s%20disease&gclid=CjwKCAjwh5qLBhALEiwAloods9Nez7LkaBBZbGqc2SMv-rsXKgmVqrmD84SoRt5op-KG-8ABkBoIBoCxakQAvD_BwE)

<sup>6</sup> Vasconcellos LF, Pereira JS. **Parkinson's disease dementia: Diagnostic criteria and risk factor review.** *J Clin Exp Neuropsychol.* 2015;37(9):988-93.

U.S. v. Robert T. Brockman  
*The Forensic Panel – Marc Agronin, M.D.*  
 October 29, 2021

Page 17 of 22

These recurrent bouts of delirium are a major diagnostic and prognostic indicator of dementia for Mr. Brockman and get to the heart of the limitations in his mental capacity. Consider these definitions offered by Fong and colleagues (including co-author Dr. Sharon Inouye, one of the world's leading experts on delirium) from a 2015 review article:

“Dementia, an insidious neurodegenerative condition, is characterised by chronic and progressive cognitive decline from a previous level of performance in one or more cognitive domains that interferes with independence in everyday activities. By contrast, delirium is a syndrome manifesting as an acute change in mental status that is characterised by inattention and disturbance in cognition that develops over a short period of time and tends to fluctuate. Delirium is a common, serious, and often fatal disorder . . . and is consistently associated with increased mortality, cognitive impairment, and functional decline.”<sup>7</sup>

Mr. Brockman's clinical history, presentation and neuropsychological profile have shown evidence of both disorders – chronic and progressive significant cognitive decline across multiple domains (e.g., learning and memory, executive functions, daily function, etc.) that has endured across all examinations, coupled with more acute changes with fluctuating symptoms of worsening confusion, inattention and disorganized thinking. In my current interview, I found Mr. Brockman to be slightly more attentive and able to stay on track and reality-based during the interview, although with a similar if not worse degree of cognitive impairment in terms of disorientation, executive / abstract thinking, memory, insight and judgment.

Although he did not manifest as delirious to me during the interview, and did not manifest as delirious in any of the other taped interviews I viewed, one cannot absolutely rule out residual delirium by virtue of documented delirium in September, due to its waxing and waning nature. Poor as his performance was, I may on this occasion have been seeing him at his best, with his worst showing exhibiting more impairment attributable to a wave of delirium.

The recurrence of delirium in the setting of infection is an important indicator of underlying dementia, and a poor prognosticator for ongoing cognitive decline. As Fong and colleagues state:

“Delirium and dementia can commonly coexist, with pre-existing dementia being a leading risk factor for delirium. . . Large cohort studies suggest that cognitive impairment and dementia are substantial risk factors for delirium. . . Careful follow-up studies have documented that persons with dementia who develop delirium have worse outcomes than those with dementia alone,

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<sup>7</sup> Fong TG, Davis D, Growdon ME, Albuquerque A, Inouye SK. **The interface between delirium and dementia in elderly adults** [published correction appears in *Lancet Neurol.* 2015 Aug;14(8):788]. *Lancet Neurol.* 2015;14(8):823-832.

U.S. v. Robert T. Brockman  
*The Forensic Panel – Marc Agronin, M.D.*  
 October 29, 2021

Page 18 of 22

including increased rates of re-hospitalisation, institutionalisation, mortality, and subsequent cognitive decline.”<sup>1, 7, 8</sup>

Adding to one’s burden is the elevated, documented risk of permanent brain damage resulting from delirium. In Mr. Brockman’s case, this is set against a pre-existing dementia that is progressive and irreversible, and without a cure. Most devastating is the functional limitation and decline which have rendered Mr. Brockman totally dependent on 24-7 supervision and even hands-on assistance for nearly every one of his daily activities.

Put together, it is clear that although Mr. Brockman was somewhat more attentive and reality-oriented during the recent interview, it is not possible to fully rule out enduring delirium which carries with it both a fixed diagnostic indicator of his underlying dementia as well as a persistent vulnerability to future bouts of delirium which threaten to further accelerate his cognitive decline.

Mr. Brockman continues to show significant apathy associated with PD, defined by Mele and colleagues in a review article on Parkinson’s disease and apathy as characterized by “behavioural, emotional and cognitive impairment including reduced interest, reduced initiative and motivation, emotional distress and intellectual impairment.”<sup>9</sup> Apathy is a well-recognized neuropsychiatric syndrome common to many neurological diseases and incurring significant dysfunction.<sup>10</sup> For Mr. Brockman, this apathy is seen in his total lack of initiative and spontaneous efforts to organize his daily life or take interest in his legal case, aside from a few confused and impulsive attempts to go to work as if he still runs his business. Mele and colleagues report apathy in 17% to 70% of individuals with PD, and go on to state that:

In PD, apathy results in poor response to motor symptom treatment, increased healthcare spending, decreased quality of life for persons with PD and their caregivers, as well as increased risk of developing dementia and increased difficulty making decisions in day-to-day life.<sup>9,10</sup>

In their review of studies with thousands of individuals with PD and apathy, the researchers found that cognitive impairment was associated with apathy, with apathy-associated deficits in the brain’s neurotransmitter dopamine causing impairment specifically in decision-making. They also found that there are few evidence-based treatment options and thus limited evidence that apathy can be effectively managed.

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<sup>8</sup> Watt D, Koziol K, Budding D. **Delirium and confusional states**. In: Noggle C, Dean R, editors. *Disorders in Neuropsychiatry*. New York: Springer Publishing Company; 2012.

<sup>9</sup> Mele B, Van S, Holroyd-Leduc J, et al.: **Diagnosis, treatment and management of apathy in Parkinson’s disease: a scoping review**. *BMJ Open*. 2020;10(9):e037632. Published 2020 Sep 9.

<sup>10</sup> March L, Margolis RL: **Neuropsychiatric aspects of movement disorders**. In BJ Sadock, VA Sadock and P Ruiz (eds), *Kaplan & Sadock’s Comprehensive Textbook of Psychiatry*, Tenth Edition. Philadelphia PA: Wolters Kluwer 2017; pp. 540-561.



U.S. v. Robert T. Brockman  
*The Forensic Panel – Marc Agronin, M.D.*  
 October 29, 2021

Page 19 of 22

The most recent sleep study indicates ongoing obstructive sleep apnea, which is improved by the use of CPAP. There was no evidence of REM Sleep Behavior Disorder (RBD) which had been suggested early on in the course of Mr. Brockman's neurological work-ups. It is possible that the sleep study did not pick up evidence of RBD as was the case with the last study due to limitations on REM sleep, or that the condition has been improved by the use of trazodone. Nonetheless, while the presence of RBD is a common comorbid finding early on in the course of PD and related conditions, its absence in a sleep study does not have any significant bearing on the diagnosis of PDD as it is seen in only about 1/3 of those with PD.<sup>11 12</sup>

Put together, all of the clinical evidence underscores the medical certainty of significant cognitive impairment and ongoing decline that well surpasses the lesser impairment seen in mild cognitive impairment. This is completely inconsistent with a malingered state of cognitive impairment.

**2) *Is Mr. Brockman able, given the nature of the charges against him, to assist his attorneys with relevant, requested facts, dates, and specifics?***

Mr. Brockman retains little insight into the nature of his cognitive impairment and its course and causes. This was evident in the clinical interview in which he could not even tell me why his mental capacity was being examined, despite the many years of multiple clinical assessments that all indicated to him in person that he was suffering from cognitive impairment due to Parkinson's disease. As he stated in my interview with him: "Well, I have a disease which is... I don't know the name of it. It's fairly well-known to treat the kind of injuries I got going on inside me." This glaring inability is a clear indication of Mr. Brockman's memory loss, poor insight and lack of detailed understanding of this and other defining aspects of his current life.

Furthermore, his inability to have a clear and detailed recall, understanding, and insight into the most fundamental of his physical and cognitive limitations that affect him on a daily basis (and even is pushed aside and contradicted in his mind when he attempts to pack his briefcase and head to his "office" in "Houston," oblivious to the fact that he no longer works and is already in Houston) is one of the strongest and most obvious indicators of his inability to provide relevant, and requested information to his attorneys with respect to his legal situation.

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<sup>11</sup> St Louis EK, Boeve AR, Boeve BF. **REM Sleep Behavior Disorder in Parkinson's Disease and Other Synucleinopathies.** *Mov Disord.* 2017 May;32(5):645-658.

<sup>12</sup> Jozwiak N, Postuma RB, Montplaisir J, Let al. **REM Sleep Behavior Disorder and Cognitive Impairment in Parkinson's Disease.** *Sleep.* 2017 Aug 1;40(8):zsx101.

U.S. v. Robert T. Brockman  
*The Forensic Panel – Marc Agronin, M.D.*  
October 29, 2021

Page 20 of 22

The re-evaluation continues to show all of the same cognitive limitations from his July evaluation, and to manifest persistent and progressively worsening cognitive impairment over the past few years. A number of these cognitive limitations reflect that Mr. Brockman does not have the mental capacity to adequately assist his counsel in defending his case.

Specifically, Mr. Brockman is limited by:

- Significant and pervasive memory lapses for names, events, and decisions relevant to the case;
- Lack of understanding of advice, suggestions and strategy provided by counsel;
- Disorientation and confusion over the timeline of events and discussions relevant to the case;
- Lack of certainty or reliability of his recollections;
- Inability to reason;
- Lack of initiation in discussing and reviewing issues relevant to the case;
- Inability to work with technology to communicate on the case;
- Lack of reliable decision-making rendering it impossible for attorneys to pursue strategic decisions that may not be abruptly reversed by him.

His underlying condition will only worsen over time, as has been revealed to date, such that his current limitations will continue to render him unable to adequately assist his attorneys. The presence of delirium has only further worsened this already incapacitated state; the waxing and waning or even recovery from delirium will not change that fundamental state of incapacity caused by his progressive and irreversible dementia.

***3) Does Mr. Brockman reflect the mental stamina needed for a courtroom trial on the charges he faces? Why or why not?***

Mr. Brockman suffers from multiple underlying cognitive and physical conditions including PD, PDD and apathy which at baseline continue to severely limit the mental stamina that would be needed to assist his attorneys and adequately participate in a courtroom trial.

Neuropsychological testing continues to show the slowing of his mental processing speed which underlies these limitations. Mr. Brockman can be quite sociable at times and demonstrates his baseline, overlearned robust language and social skills. However, even these relative cognitive strengths have waned, with him lapsing into digressions that are nonsensical, impulsive and even inappropriate (such as speaking to an acquaintance about a confabulated aspect of his sex life).

Mr. Brockman is also showing significant progression of his physical frailty, with several recent falls resulting in minor injury. He was noted to be quite unsteady on his feet by his caregiver and wife, and hesitant in his movements during my exam.

U.S. v. Robert T. Brockman  
*The Forensic Panel – Marc Agronin, M.D.*  
 October 29, 2021

Page 21 of 22

**4) *Is Mr. Brockman able to assist his counsel in defending his case? Why or why not?***

During his adult life, Mr. Brockman has been, at baseline, an extremely intelligent, sociable and loquacious individual with the “gift of gab.” He has also been described as a corporate leader who always took a strong and paternalistic interest in his family, friends, and co-workers, building an incredibly successful company in the process. The man who presents through all clinical interviews, however, both with the government-consulting examiners and each of The Forensic Panel’s specialists, is a shell of this man and declining by the day.

Though he is able to reply to questions posed to him in a generally cooperative and jovial manner, a close read of his responses in both interviews and neuropsychological testing demonstrates a man with a passive demeanor, a vague and disorganized response set devoid of insight and good judgment, slowed and confused mental processing and reasoning, and a form of thinking that easily digresses into irrelevant rabbit holes of his previous work. This state of mind is a direct result of Parkinson’s disease dementia which is being further degraded by the build-up up the neurotoxic amyloid protein seen in Alzheimer’s disease, and weighed down by severe apathy and recurrent bouts of delirium.

An overwhelming amount of clinical evidence confirms his dementia diagnosis and associated cognitive impairment, derived from in-person assessments from multiple independent examiners and supported by both repeated standardized neuropsychological testing and physical biomarkers in the form of neuro-imaging studies. All of this data provides clinical and forensic explanation as to why Mr. Brockman does not have the mental capacity to adequately assist his counsel in defending his case.

- He does not and cannot have the motivation to actively ponder his case and initiate contact with his attorneys on case-related matters due to disorientation, lack of insight and apathy;
- He does not and cannot recall sufficient details in context when called upon from his attorneys due to short-term memory impairment and waning access to previous memories;
- He does not and cannot have the ability to sufficiently weigh the details of his case, understand the legal predicament or strategy and reason about it in order to actively make decisions and guide his attorneys in a meaningful way;
- Though he understands to some extent the gravity of his legal situation, he cannot and does not have the mental ability and acuity, memory recall, learning ability, and judgment to actively engage in logical and meaningful discussions with his attorneys;

U.S. v. Robert T. Brockman  
*The Forensic Panel – Marc Agronin, M.D.*  
October 29, 2021

Page 22 of 22

- He does not and cannot utilize technology or memory supports (emails, texts, phone calls, notes) in order to initiate or respond to communication from his attorneys as a result of his disorientation, apathy, impaired executive function and praxis, and remains totally dependent on others to do this for him;
- He does not and cannot take ownership of an engaged and meaningful role in his own defense due to his impaired motivation, insight, judgment and executive abilities; instead he responds passively to all questions with vague answers that are often influenced by the nature of the question or extraneous information, such as irrelevant aspects of his previous work. As a result, the integrity of his knowledge about the case is fractured, confabulated and often just plain wrong, rendering his responses nonsensical, irrelevant and false.

This state of incapacity is permanent and progressive, extremely vulnerable to future damage to his brain through infection and injury, and there do not exist any treatments to reverse or, at this point, even mitigate its effects.

Very truly yours,

A handwritten signature in black ink, appearing to read 'M. Agronin' with a stylized flourish at the end.

**Marc E. Agronin, MD, DFAPA, DFAAGP**

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